EXPERIMENTAL MILIARIA IN MAN

V. The Effect of Poral Closure on the Secretory Function of the Eccrine Sweat Gland*

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In previous papers of this series (1–4) it has been shown that a variety of minor epidermal injuries may induce keratotic closure of the pore of the eccrine sweat gland. Such poral occlusion always results in anhidrosis of the affected glands.

Although the capacity to form sweat is unchanged, any sweat secreted cannot escape to the skin surface. Actually it can be demonstrated that under these circumstances the entrapped sweat may rupture the sweat duct at various levels producing different clinical signs of sweat retention. It can be shown from physiologic and histologic studies that in the event of rupture, sweat continues to form and be absorbed at the site of vesiculation. However, in the absence of sweat duct rupture, it is not known whether or not the secretory tubules continue to function. One view is that sweating does continue and that the sweat is reabsorbed by the ductal epithelium or that it diffuses through the intraepidermal ductal segment. The other view holds that sweat formation is stopped by the increased intraluminal pressure.

The present study, the final in this series, was undertaken to determine the functional status of the eccrine sweat gland in the presence of poral blockage. Keratotic occlusion of the sweat pores was achieved by the application of aluminum chloride solution, the glands were stimulated and tubular glycogen levels were determined histochemically. In the presence of continued active secretion, the glycogen disappears from the gland (5).

METHOD

Twelve normal healthy male volunteers served as subjects. In each of these, two anhidrotic patches (sites A, B) were produced on the back by the application of a closed 48-hour patch test of a 20% aqueous solution of aluminum chloride. Immediately after removing the patch tests, 1 cc. of a 1/1000 aqueous solution of atropine sulfate was injected into anhidrotic site A, and 1 cc. of physiologic saline was injected into anhidrotic site B. Thereafter, the subjects entered a heating chamber (1) which stimulated profuse generalized sweating. Inspection revealed sites A and B to be anhidrotic and to present no clinical changes in 10 of the 12 men. In 2 of the men minute papulovesicles could be seen in site B, whereas site A remained normal. Biopsies (5 mm.) were taken, after 30 minutes of sweating. No anesthetic was employed since a high speed rotary punch was used (6). The biopsies were fixed in Rossman's solution and

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sections were stained alternately with hematoxylin and eosin, and the McManus procedure for glycogen (7).

RESULTS

In all ten of the biopsy pairs in which clinical changes were absent there was abundant glycogen to be found in the tubular secretory cells of both the atropin-

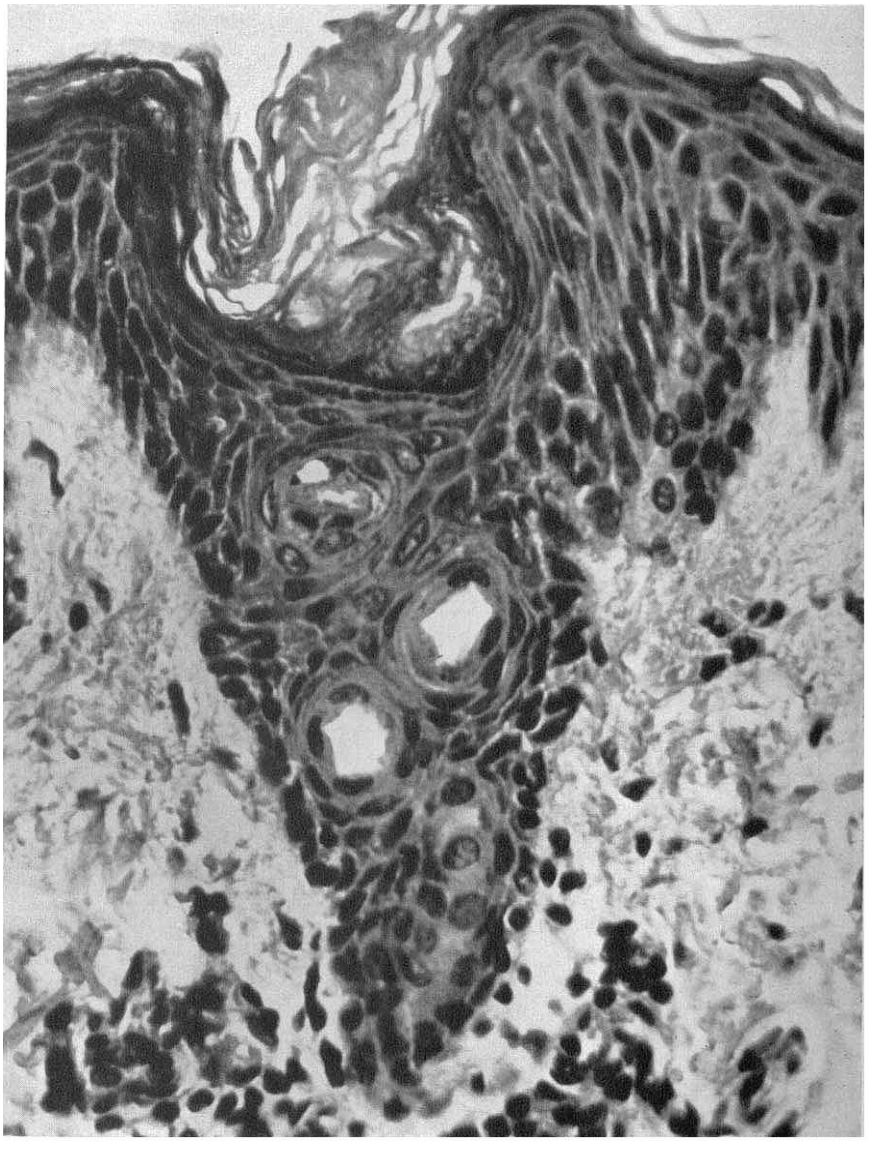

FIG. 1. Sweat retention anhidrosis with no clinical changes. View of terminal eccrine sweat duct showing keratotic plug (1000 ×). Note duct is dilated but periductal epidermis is normal. No rupture has occurred. Adjacent section showed normal glycogen level in tubular cells. Biopsy specimen from site B.

ized control anhidrotic glands (site A) and in the anhidrotic glands of site B. Histologically keratotic plugging could be seen in the orifices of these sweat glands, but no evidence of rupture was present (Fig. 1).

In two specimens taken from site B in which papulovesicles developed during exposure to heat, glycogen was absent from the tubules whereas in the control

FIG. 2. Sweat retention anhidrosis with associated papulo-vesiculation. View of ruptured dilated terminal eccrine sweat duct showing keratotic plug (1000 ×). Note epidermal disruption in tip of peg, evidence of extravasation of sweat. McManus stains revealed absence of glycogen in tubular cells supporting view that sweat continued to be formed.

atropinized glands of the normal appearing site A glycogen was present in considerable quantity.

Histologically keratotic plugging could be seen in the specimens from sites A and B, and in the two site B biopsies, definite evidence of ductal rupture could be seen (Fig. 2).

DISCUSSION

The glycogen content of the tubular secretory cells accurately reflects the recent functional status of these cells. In the presence of active secretion over a period of 30 minutes to an hour, the glycogen disappears. In the normal resting eccrine gland glycogen is always present. Employing this histochemical index of secretory activity we are able to conclude that sweat is *not* secreted, in other than minimal quantities, by an intact yet obstructed gland. It would appear that the normal ductal epithelium is incapable of absorption of appreciable amounts of sweat and that accordingly intraluminal pressure rapidly rises with a subsequent secretory standstill. However, if the intraluminal pressure leads to rupture of the duct, as revealed by clinical changes, sweat continues to be produced in normal quantities and it escapes into the epidermis or dermis at the point of rupture. This "internal sweating" is evidenced not only by the histologic changes previously described, but also by the histochemical changes in glycogen level.

The use of atropine in the control sites A afforded a method of pharmacologically blocking all sweat gland activity at the adenoneural junction. Thus, none of the atropinized glands showed any secretory activity in response to heat, and their glycogen content represented true resting levels for comparison with the levels seen in glands rendered anhidrotic as a result of poral closure. Furthermore, biopsies of sites A proved that the aluminum chloride applications had had no effect on the secretory epithelium.

SUMMARY

Experimental eccrine sweat retention anhidrosis was produced by the application of aluminum chloride. Studies were made of the glycogen levels of the tubular secretory cells as an index of secretory activity. It was concluded that the sweat gland shows minimal activity in the presence of keratotic plugging of the pore, unless rupture of the duct occurs. In the event of rupture, sweating can be maintained at normal rates, as evidenced by the rapid disappearance of glycogen from the tubular secretory cells.

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